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SOME ESSENTIALS FOR SATISFACTORY WORK IN ALLERGY*

J. H. BLACK

Dallas, Texas

Within recent years it has been shown conclusively that (1) allergic conditions are quite frequent and are found in a considerable part of the population, and (2) that many individuals may be relieved by proper diagnosis and treatment. This has led to a marked revival of interest in these conditions and many men have already entered or are contemplating entrance into this type of work. This, I believe, justifies setting forth a few well-defined essentials which should be understood by those undertaking the care of such persons. There are, of course, a vast number of technical details and bits of information that one acquires in this work which serve to increase the skill and knowledge of the worker. Many of these may be learned by perusal of the literature. I wish to call to your attention in this discussion a few necessities which must be acquired before satisfactory work may be done.

First essential: Ability to differentiate between allergic and non-allergic conditions.

It should be self-evident that treatment on an allergic hypothesis of a non-allergic person is useless. But it should be pointed out that differentiation is not always easy. Patients not infrequently present themselves with a diagnosis of bronchial asthma whose dyspnea is due to cardiac weakness. It is not always easy to determine whether a patient is suffering from a hay fever or an acute coryza. A child was sent to me with a diagnosis of an allergic nasal condition which proved to be a leutic periostitis of the nasal bones. One must know as much as possible of general

* Read before the Eleventh Annual Convention of the American Society of Clinical Pathologists, New Orleans, Louisiana, May 6–9, 1932.

medicine in order to avoid mistakes of this kind. A good practice, I think, is to consider every patient non-allergic until he is proved to be otherwise. This may cause the loss of an occasional patient but it will prevent some unhappy moments.

Second essential: Some knowledge of the mechanism underlying allergic manifestations.

Even a casual survey of the literature will convince one that a vast amount of knowledge is lacking but there are certain facts and theories which form a basis for this study and ignorance of these leaves one carrying on his work empirically and with no rational basis on which to build. Every one engaged in this work should be able to contribute something to its growth by his own observations. It cannot be done by one who has acquired only the technical skill for the necessary procedures.

Third essential: A working knowledge of the various substances which may act as allergens.

It certainly is important to know the botany of the locality in which one works. One must know what grasses, weeds and trees are found, their relative abundance, the time of pollination and their botanical relationships. The five postulates of Thommen should be kept in mind: (1) the pollen must contain an allergen, (2) the pollen must be wind-borne, (3) the pollen must be produced in sufficient quantity, (4) the pollen must be sufficiently buoyant to be carried a long distance, (5) the plant producing the pollen must be widely and abundantly distributed. Lack of knowledge regarding these factors makes for failure in treatment.

Environmental substances, some of which are occupational dusts, must be known. Animal emanations and dusts of various kinds occur under many and diverse circumstances and the more information one has regarding these substances the better the results of treatment.

Even a knowledge of foods and their preparation is a prime requisite. A patient may be so sensitive to egg white as to react to the small amount found in most baking powders. If one does not know that baking powders commonly contain egg white, failure with this type of patient may result. The removal of a given substance from the dietary is not complete until all foods

containing even a small amount of the substance are removed. Unless one knows something of the composition and preparation of foods, good results will be infrequent.

Fourth essential: Willingness to devote time to a painstaking history and examination.

In no other field of medicine is this more important. Allergy is not a special isolated phenomenon which can be cared for without regard for the rest of the patient. Allergic conditions are definitely affected by the psychic state, endocrine disturbances and autonomic nervous system reactions. A careful investigation into all the possible interrelationships may be necessary and nothing is of more value than a detailed history. In many instances the allergen may be discovered only by good "detective work" and infinite patience is a prerequisite.

Fifth essential: Ability to interpret skin reactions.

There has been the impression, fostered by some commercial institutions, that the diagnosis of the etiologic factor in allergy may be made easily and certainly by skin tests. This, unfortunately, is frequently untrue. Skin reactions are a valuable aid in diagnosis but they are subject to error and slavish dependance upon them will often lead one astray. Reaction to a given substance may occur in the absence of clinical sensitiveness to that substance or be absent in spite of definite clinical sensitiveness. Skin reactions should be correlated with the patient's history and knowledge of his environmental and dietary relationships. A young physician who had examined a patient with hay fever asked me to supply him with two pollens for treatment. One of them was a pollen which is believed never to cause hay fever, the other was one found in the air at a season entirely different from that in which the patient had his symptoms. Both of these pollens may have reacted but some other pollen caused the hay fever of the patient.

There are advocates of both scratch and intradermal testing. Because of the danger of severe reactions it is my custom to use intradermal tests only after scratch tests have been made and found negative. A positive reaction to scratch testing does not need corroboration by intradermal technic. A negative reaction

by scratch test may be checked by intradermal injection with safety. This method causes some duplication of effort but it is both efficient and safe. Scratch tests are frequently not effective and intradermal tests, without preliminary scratch tests, may be unsafe. The character and amount of reaction may differ considerably with the scratch and intradermal technic. Considerable experience with both methods and careful observation are necessary to evaluate reactions.

Sixth essential: Recognition of multiple sensitization as the rule rather than the exception.

In the past many of us have been guilty of examining patients sufficiently to find one substance reacting which might be correlated with the history and stopping all diagnostic effort at that point. The knowledge has been forced on us that allergic individuals are usually sensitive to more than one thing and they have the tendency to become sensitive to other things which may become of clinical importance if the exposure of the patient to the allergen is sufficient. The examination should be inclusive enough to cover all possible factors relating to the condition which the patient presents.

Seventh essential: Potent extracts which deteriorate slowly must be used, some definite standard of measurement must be adopted and, in treatment, the dosage must be individualized.

There are various methods of preparation of extracts. Of these, various modifications of a glycerin saline solvent are most generally used. The preparation of extracts is quite simple and satisfactory solutions may be purchased or made.

There is no certainty at present as to the chemical nature of the allergenic substance so there is no entirely satisfactory standardization. The three methods in use are based on the nitrogen content of the extract, the amount of allergen in the solution expressed in units, and the amount of allergen in the solution expressed in terms of percentage of solute to solvent. Any one of these methods may be used if the user is acquainted with it and recognizes its limitations.

It should be perfectly evident that treatment must be individualized and routine dosage cannot be followed. Some patients

require much smaller increments in dosage than others and some require that treatment be carried to a much higher level to obtain protection than do others. Unthinking adherence to a printed schedule of dosage may lead to failure or over treatment.

Eighth essential: Knowlecge of action and dosage of various medicinal agents.

In addition to specific treatment of allergy the management of patients in acute attacks and the use of non-specific agents in the control of the allergic state are important.

It is important to know that epinephrin may be used in large doses and that morphine may be very dangerous, that children frequently require epinephrin in doses as large as those given to adults and that some drugs which may be effective in relieving attacks may be contraindicated because of a hypersensitiveness to the drug. Many details, which may not need consideration in other conditions, do demand attention and the ability to use properly the various medicinal agents is essential to the comfort and even the safety of the patient.

Ninth essential: It is important to know how to advise patients so they may secure good results and to know what may be promised in the way of cure.

While the results of accurate diagnosis and careful treatment have improved year by year, it is evident that the intelligent, continued coöperation of the patient is necessary and this is best secured by a frank, full discussion of treatment and what may be expected from it. It is further evident that whether the patient may be promised prompt and complete relief, delayed but sure cure, or only a partial amelioration of symptoms depends upon several factors which must be known if a correct prognosis is to be made. Many patients are disappointed because of over-enthusiasm and lack of knowledge which cause the physician to promise more than he can do.

Tenth essential: Great patience on the part of both physician and patient.

Treatment of many allergic individuals is not a matter of days but of months and even years. Even after the patient has been told of the long, continued treatment required, a certain number

become discouraged and need support. A physician who wants to see results at once and who cannot possess his soul in patience had better shun work in this field. A friend once said to me that no one would do this type of work but an old maid. Whether the terminology is correct or not the idea of meticulous attention to details and continued effort along the same line is correct.

These ten essentials by no means exhaust the list of requirements for satisfactory work in allergy. There are multitudinous details which go to make up the art and science of care of allergic patients. But the points discussed are necessary if one is to undertake this work, in a competent manner. Satisfactory work in allergy does not require any unusual order of intelligence but it does demand an enthusiasm and willingness to work which will carry one over a good many disappointing days which are sure to appear.

MYELOID IMMATURITY IN PERNICIOUS ANEMIA*

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In the morphologic study of pernicious anemia the macrocytosis of the erythrocytes plays such a striking part that relatively little attention is paid the leukocytes, other than to note leukopenia or relative lymphocytosis. Next in significance to the macrocytosis is the presence of hypersegmented polymorphonuclear neutrophils, or neutrophils showing pathologic hyperpolymorphism. Besides these changes from the normal, there may occur, particularly in the severer degrees of anemia, immaturity in cells of the myeloid line back to the stem cell or myeloblast. Although the number of immature cells is seldom great enough to throw doubt on the diagnosis, occasionally a case will be seen in which the immaturity is so marked that, at least temporarily, a diagnosis of myelogenous leukemia is suggested. The observation of such a case resulted in this study of the incidence of myeloid immaturity in a series of sixty-five cases of pernicious anemia.

The literature contains a number of reports on this point, but in many of them the data are incomplete and determinations of hemoglobin or erythrocyte or leukocyte counts are not given. Ziegler[10] reported a case in which there was 4 per cent neutrophilic myelocytes, which increased to 14 per cent following infusion of salt. Naegeli[6] stated: "I find myelocytes in the majority of cases ($\frac{1}{2}$ to about $1\frac{1}{2}$ per cent). Their occurrence is a capricious one. According to former theories, they are signs of severe marrow affection and signify in no way the beginning of a myeloid hyperactivity." In the severer degrees of anemia, Zadek[9] found myelocytes present more frequently than absent, and in his study of 200 cases he was able to demonstrate as high as 6 per cent

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myelocytes without necessarily having an increase in the percentage of metamyelocytes above that of the myelocytes. Eight of twenty-two cases studied by Hittmair[4] showed myelocytes; in five of the eight there were promyelocytes and in four, myeloblasts. Schauman and Saltzman[8] found normal myeloblasts in 50 per cent of their cases but only at the height of the disease, and they also mentioned the transitory nature of these observations. Anderson[1] stated that Downey found myeloblasts in a case he reported. Neuburger,[7] Jedlička and Beránek,[5] and Held[3] among others reported on the immaturity. Arneth,[2] however, vigorously denied their presence and stated that in his cases even at the height of the disease and just before death, myelocytes could not be demonstrated.*

Zadek rightly emphasized that in considering the morphologic picture of pernicious anemia at any particular time, one must take into consideration whether the patient is in beginning relapse, at the low point of any particular relapse, or in beginning remission, and must consider the rapidity of remission, since all these affect in varying degree, the blood picture from the standpoint of leukocytes. This also holds true in the study of the erythrocytes, for in the very early stages of pernicious anemia or in the late stages with the disease in an aplastic phase, macrocytosis may be absent.

The material for this study was selected at random by the laboratory technician, without regard for the differential count, but a larger number of cases among the more anemic group were examined. The differential count was made on 500 cells, this number being arbitrarily chosen. With a few exceptions I made all determinations. The table shows that it is likely in the ordinary differential count of 200 cells, immaturity might easily have been missed in some cases. By the same token it would be fair to assume that some of the cases without immaturity would have shown it if 1,000 or 2,000 cells had been counted. The terminology used is that of Pappenheim, so that myelocytes and

* N. Rosenthal has called my attention to a case reported by Brill (Tr. Assn. Am. Phys., 30: 547–561. 1915) in which, following aplenectomy for pernicious anemia, leukocytosis of 73,000 developed with as high as 11.9 per cent neutrophilic myelocytes and 8.5 per cent myeloblasts.

promyelocytes correspond to the mature and immature myelocyte of other authors, whereas leukoblast and myeloblast are both included under the heading of stem cell or myeloblast by the same authors.

For purposes of classification cases have not been considered as showing immaturity unless myelocytes were seen. On this basis twenty-five of the sixty-five cases (including one case reported in greater detail later) fall into this group; in nineteen of these leukoblasts or myeloblasts were demonstrated; in five promyelocytes, and in one case only, myelocytes. In some instances

TABLE 1

DIFFERENTIAL BLOOD COUNT IN A CASE OF PERNICIOUS ANEMIA

	1-6-30	1-14-30	1-15-30	1-16-30	1-17-30	1-18-30	1-20-30	1-21-30
Hemoglobin*	22	28		30		40		40
Erythrocytes†	1.05	1.39		1.64		1.85		2.07
Leukocytes	3,100	22,600	11,500	9,200	6,200	5,000	8,800	7,100
Neutrophiles	75.8	37.2	47.8	64.8	66.0	66.0	77.2	82.4
Lymphocytes	20.8	9.0	9.0	6.4	10.6	14.8	16.2	10.4
Monocytes	1.8	5.2	11.0	9.2	12.6	7.4	2.8	4.4
Eosinophiles	1.2	5.2	6.8	4.8	5.4	7.4	2.6	2.8
Basophiles		0.4		0.2		1.2	0.2	
Metamyelocytes		14.2	8.2	8.4	1.6	0.6		
Myelocytes		11.6	10.0	2.8	2.0	2.0	0.8	
Promyelocytes	0.2	13.0	4.6	3.4	1.8	0.6	0.2	
Leukoblasts	0.2	3.6	2.0					
Myeloblasts		0.6	0.6					
Reticulocytes	0.1	14.2		19.4	21.4	10.6	9.3	11.2
Volume index	0.99	1.31		1.33				
Blood urea‡	72§	38		30	28	32	24	

* In per cent. † In millions. ‡ Mgm. per 100 cc. § 1-9-30.

only one or two of the more immature forms could be found, and in others they were present in relatively generous numbers. Leukoblasts or stem cells were not found in any case with an erythrocyte count higher than 2,030,000, but in fifteen cases in which erythrocyte counts were lower than this, immaturity could not be found. The point emphasized by both Neuburger and Zadek that metamyelocytes may not be found in as large numbers as myelocytes or promyelocytes is supported by my observations. Four cases have been studied at intervals of one to four days to

determine how rapidly the immature cells disappear from the peripheral circulation when the patient is placed on treatment of one form or another. In two cases in which intravenous injection of liver extract was employed all immaturity had disappeared by the nineteenth day. When oral treatment was used the immaturity disappeared within approximately the same time (the twentieth day). After the institution of treatment there appears to be a temporary increase in the amount of immaturity, but this rapidly disappears with clinical improvement.

Because of the unusually marked reaction in one case, a table of details has been arranged (table 1).

REPORT OF CASE

A man, aged fifty-four years, came to The Mayo Clinic January 3, 1930 complaining of weakness and loss of sensation in his feet. In February, 1929, he had consulted his home physician because of swelling of feet and hands associated with numbness and tingling, intermittent diarrhea and sore tongue. His case had been diagnosed as pernicious anemia and liver extract and whole liver had been prescribed. Improvement followed, and during the summer of 1929 the erythrocyte count was almost normal. Failure to carry out the diet conscientiously resulted in relapse of symptoms.

The skin was pale and lemon yellow. The tongue was smooth and atrophic. The spleen was definitely palpable. There was slight edema of the hands and feet. The blood pressure in millimeters of mercury was 158 systolic and 106 diastolic; the pulse rate was 82 beats each minute and the temperature 97°F.

The patient was sent to the hospital and January 4, 1930, the concentration of hemoglobin was 20 per cent, erythrocytes numbered 1,010,000, and leukocytes 1,800 in each cubic millimeter of blood. Free hydrochloric acid could not be demonstrated in the gastric contents at the end of one and a fourth hours. Urinalysis was negative. Roentgenograms of the thorax January 4 and 9 gave negative results. Fluoroscopic examination of the stomach was negative. January 7 he was placed on a diet containing ½ pound of raw swine stomach each day. During his stay in the hospital his temperature varied from normal to 100.6°F. and January 8 he was delirious most of the day. The concentration of urea at this time was 64 mgm. in each 100 cc. of blood. Five hundred cubic centimeters of 10 per cent glucose in 1 per cent saline solution was given intravenously and this was followed January 9, 1930, by a transfusion of 500 cc. of whole citrated blood which caused a chill and rise in axillary temperature to 102°F. On this date the concentration of urea rose to 72 mgm. in each 100 cc. of blood. The urine contained pus graded 2, and 60 cells to the field. This was the only time during the patient's stay in the hospital that the urine was found to be abnormal. January 8 the treatment had been changed to Lilly's liver extract 343, four vials being given on that day; this was increased to eight

vials on subsequent days. A rapid drop in the retention of urea occurred, so that by January 13 there was only 32 mgm. of urea in each 100 cc. of blood. After this date the concentration of urea on one occasion reached 38 mgm., but at the time of the patient's dismissal January 29 it was 24 mgm. in each 100 cc. of blood.

An infectious process could not be found to account for the slight fever, and after January 16 the temperature never rose above 99°F. The thorax, abdomen and upper part of the respiratory system did not afford positive data, and at the height of the leukocytosis, January 14, a roentgenogram of the thorax showed it to be normal. From January 10 on there was rapid improvement in symptoms and January 20 the liver extract was reduced to six vials a day.

The first blood smear was examined January 6. Morphologically there was no macrocytosis, although the cells were well filled with hemoglobin. This is in agreement with a volume index of 0.99 on that day. The neutrophils, however, showed increased segmentation. There was little evidence of regeneration in the erythrocyte line and five counts of reticulated erythrocytes from January 4 to 10 showed a maximal of 0.1 per cent of these cells. The next smear examined was taken January 14, at the height of the leukocytosis. By this time there was marked macrocytosis with many polychromatophilic cells. The numerous immature cells gave the appearance of chronic myelogenous leukemia, although the macrocytosis was so marked that there seemed to be little doubt about the diagnosis. The increased segmentation in the neutrophils persisted during this time. January 13 and 16, volume index determinations were 1.31 and 1.33, respectively, so that this conformed to the change in appearance of erythrocytes. The rapid return to normal differential count corresponded to the clinical improvement.

Eight days elapsed between examination of the first two smears, and it is possible that the maximal reaction may have been missed. Since on January 11 the leukocytes numbered only 2,500 it is unlikely that there was ever much more marked reaction than on January 14. What part the retention of urea in the blood played in bringing about this reaction, if any, is difficult to determine.

Zadek reported that in a rapidly occurring spontaneous remission in pernicious anemia he had observed a leukocyte count of 20,000 with an increase in immaturity at that time. Remissions in pernicious anemia induced by modern methods of treatment are probably essentially not different from those occurring spontaneously when considered from a morphologic standpoint except for the more rapid recovery and return to a much higher hemoglobin and erythrocyte level than usually occurs spontaneously.

In the morphologic differential diagnosis an occasional case of early acute or chronic leukemia may be seen in which there is mild hyperchromatic macrocytosis, and in which the amount of

immaturity has not yet reached a degree sufficient to make a positive diagnosis. If the patient is suffering from pernicious anemia, adequate treatment will soon cause disappearance of the immaturity instead of the increase which would occur in the usual course of acute or chronic leukemia.

SUMMARY

In nineteen of sixty-five cases of pernicious anemia, leukoblasts or stem cells could be demonstrated in a differential count of 500 cells; five cases showed promyelocytes and in one case only myelocytes were found.

As a rule immaturity occurs primarily in cases in which erythrocytes are below, 2,000,000, although in fifteen cases in which they were below this point immaturity was not found.

After the institution of modern treatment there may be an increase in the amount of immaturity within a short time. This is occasionally quite marked.

In the four cases observed for a considerable period, the immaturity had disappeared by the twentieth day.

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THE HEMATOPOIETIC SYSTEM AND INFECTION*

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Every pathological process, to be understood, must be considered from its physiological source. In discussing leukemias or any of the so-called blood diseases we must consider the normal or physiological process with which the blood cells and the hematopoetic system are concerned.

All blood cells are derived from a stem cell belonging to the reticulo-endothelial system which is found all over the body, particularly in the bone marrow, liver, spleen and lymph nodes. The erythrocytes and granulocytes (leukocytes) are formed in the bone marrow, the agranulocyte (lymphocyte) is formed in the lymph system, and still a third white cell which is neither granular nor agranular, called the monocyte, has its origin in the reticulum cells of many organs. (See fig. 1.)

These cells are constantly being produced by their respective formative tissues, function in their individual capacities, and are then destroyed. There is therefore a constant and perpetual production and destruction of blood cells occurring in the body as a physiological process. While some of these cells are probably formed in the reticulo-endothelial system of the spleen, it is this organ together with the lungs which is particularly concerned with destruction of the aged cells. It is quite conceivable that these two processes, the manufacture and destruction of leukocytes, are so related as to balance each other and maintain an equilibrium which is compatible with health. If there is a body demand, such as occurs in infection, for an increased number of leukocytes the production process is stimulated and the formative organs expel a greater number of such cells into the peripheral

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circulation as evidenced by an increased leukocyte count. After the demand is met the destructive process is stimulated, the leukocytes in excess are destroyed and the normal equilibrium between these two processes is again established as evidenced by reduction of leukocytes to within normal limits. Similarly some such mechanism must be operative in the maintenance of a normal number of erythrocytes in the peripheral circulation.

Normally the leukocytes are subject to only slight and temporary variation in number such as may be caused by muscular exertion or digestion. With the introduction however of any foreign substance into the body, as illustrated by infection, the forces representing the body defense mechanism are immediately stimulated. Foremost among these forces is the reticulo-endothelial system which supplies the body demand by an increased supply of leukocytes or other cells needed to combat the interference with the normal cell balance. The blood changes in pathological conditions may, on this basis, be due to the same processes of production and destruction as occur in normal metabolism but in an exaggerated form. That is, an increased leukocyte count in disease may be explained as resulting from an increased cell formation accompanied by a proportional decreased cell destruction.

The pathological condition underlying any of the so-called blood diseases is found in the blood forming elements and not in the blood; we must then consider aplastic anemia and agranulocytosis as diseases of the bone marrow, Hodgkin's disease as a disease of the lymphatic system and the leukemias as diseases of the general hematopoetic system. It is universally accepted that aplastic anemia and agranulocytosis are almost always complicated by severe gangrenous inflammations located on some mucous surface. These two conditions are in some way related and differ pathologically only in the extent of involvement found in the formative tissues. In aplastic anemia the entire hematopoetic system of the bone marrow is involved and the granulocytes, erythrocytes and blood platelets are greatly reduced in number. In agranulocytosis only the granulocytes are greatly reduced or absent while the erythrocyte count is normal or just

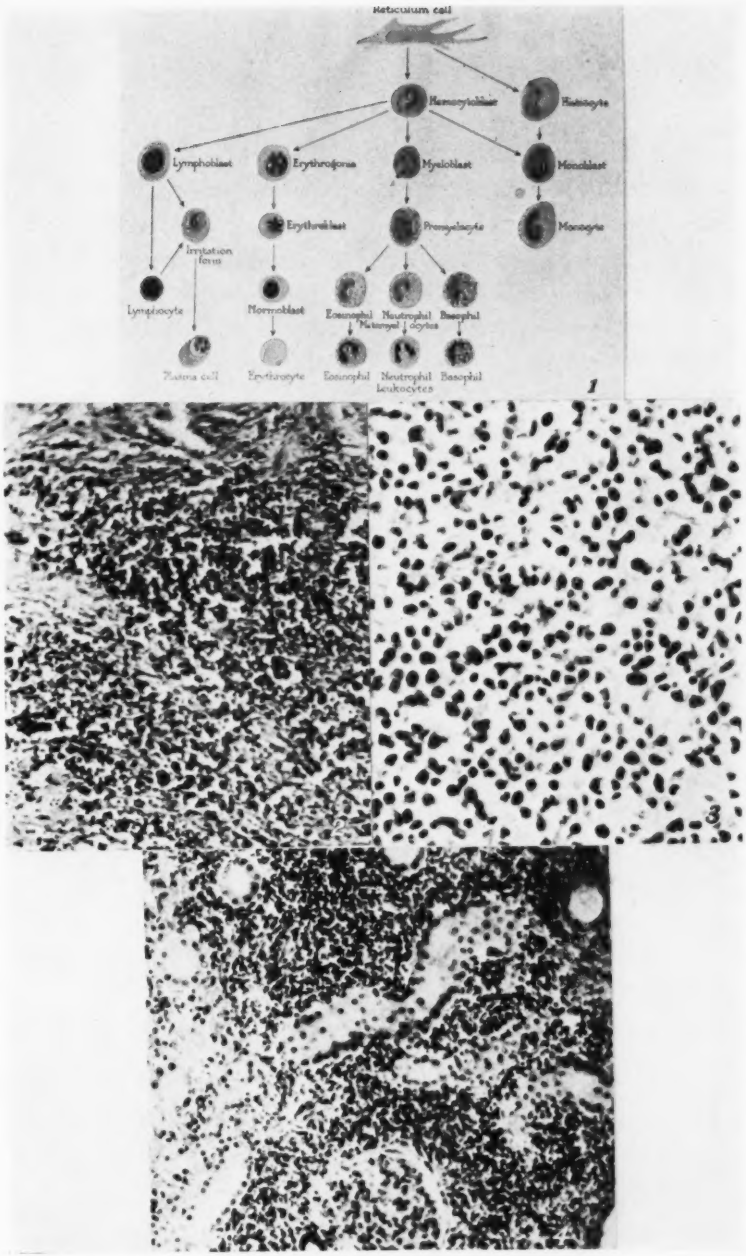


FIG. 1. NORMAL CELL DERIVATION
After R. H. Jaffé

FIG. 2. VARIED CELLS OF HODGKIN'S DISEASE

FIG. 3. MYELOGENOUS LEUKEMIA OF SPLEEN
(Various forms of granulopoesis)

FIG. 4. LYMPHATIC LEUKEMIA OF KIDNEY
Infiltrations pushing aside the tubules

below normal. In both conditions, however, gangrenous inflammations are encountered as complications. It may be assumed that some abnormal body constituent accounts for the lack of proper activation of the hematopoetic system when the body demand is increased as, for instance, in infection.

Similarly we must concede that hemorrhagic tendencies are indeed characteristic of both inflammation and some of the diseases involving the hematopoetic system. Symptomatic purpura hemorrhagica is secondary to many septic processes and is almost always found in certain types of endocarditis. Coke[2] definitely supported this theory of sepsis in purpura and Lovett[6] isolated *Pseudomonas aeruginosa* (B. pyocyaneus) from throat lesions with which he reduced the granulocyte count in rabbits.

Hodgkin's disease, variously classed between pseudo-leukemia and lymphosarcoma, presents a histological picture which varies from hyperplasia in the early stage to fibrosis in the late stage. As the disease progresses, however, a rather characteristic picture is seen: marked hyperplasia of lymphoid cells, numerous plasma cells, endothelial cells, eosinophiles and many very large multinuclear giant-cells. These cells, while not in formation of the usual inflammatory process, seem to favor infection rather than new growth as their basic origin (Fig. 2). It has long been recognized that a close relationship exists between tuberculosis and Hodgkin's disease and that a similar relationship exists between the leukemias and infection is quite plausible.

Because of the trinity idea of the three types of leukocytes, leukemias are accordingly classified into the three corresponding types: (1) myelogenous leukemia or myelosis with existing pathology in the bone marrow, (2) lymphatic leukemia or lymphadenosis with existing pathology in the lymph system, and (3) monocyte leukemia or reticulosis with existing pathology in the general reticulo-endothelial system.

In the clinical interpretation of the leukemias, there is limited evidence of any definite etiological factors. The fact that many cases of leukemia seem to begin with the sudden opening of some focus of infection favors the infectious theory of etiology. Ellerman[3] demonstrated quite clearly the infectious nature of leu-

kemia of the fowl and maintained that the causative organism is a filtrable virus. While these findings are not yet applicable to man there is considerable evidence that the leukemias are infectious in origin. Ewing[4] stated that he had seen leukemic processes develop from pneumonia and acute tonsillitis. Jaffé[5] was quite firmly convinced that leukemias are infectious in origin and cited cases of acute leukemias, especially in the young, following, the opening of a chronic focus of infection as for example by extracting an abscessed tooth. He further reported a case of a peculiar form of septicemia in which there was an active proliferation of the reticulo-endothelial cells and suggested the term spetic reticulosis.

It is rather interesting also to note that as ordinary infections produce an increase in the number of leukocytes in the peripheral circulation, meta-myelocytes and the younger forms of mature leukocytes are increased in number. The more severe the infection the younger are the mature leukocytes and in very severe infectious processes even immature cells may be seen in the peripheral circulation. It is on this basis that the various indices, such as have been described by Arneth[1] and Schilling are established. In the leukemic pictures also these younger mature forms are greatly increased in number even though the true immature cells predominate.

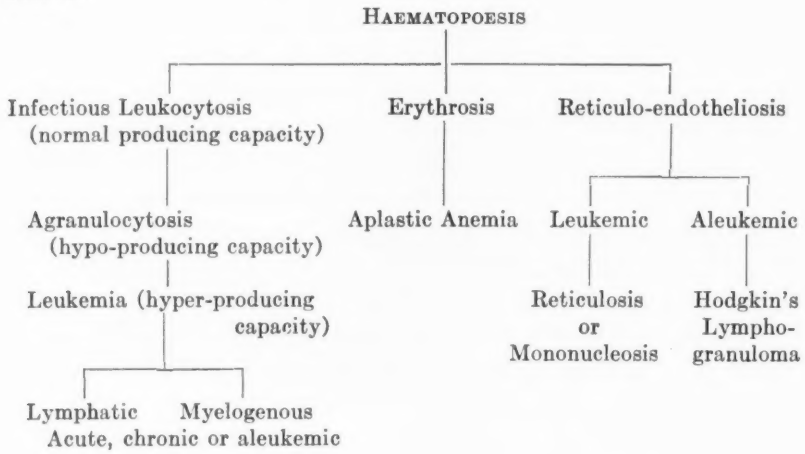
In the pathological interpretation of the leukemias as demonstrated by the microscopical examination of the hematopoetic tissue there seems to be no difference between acute and chronic forms and no essential difference between leukemia and aleukemic leukemia. In all leukemic cases whether chronic, acute or aleukemic the essential findings are marked proliferation of immature cells in either the bone marrow or the lymphatic system, and formation of immature hematopoetic tissue in the liver and spleen. An explanation of the aleukemia, offered by Szilard,[7] is that these immature cells are very fragile and break down as soon as they enter the peripheral circulation.

In a section of the spleen in myelogenous leukemia, for instance, great accumulations of granulocytes are seen (fig. 3). These cells are in various forms of immaturity (myeloblasts

to mature myelocytes) and indicate the various forms of granulopoesis. Many of these cells are large with large irregular nuclei and present a picture that may resemble that of malignant tumor but they do not have the destructive tendency of a malignant cell. They are instead the various types of immature cells sent into the periphral circulation probably because of some improper activation of the formative tissue.

Many authors are of the opinion that the chronic forms belong in the class of neoplasms, but the microscopical picture does not bear out such a contention. Although these cells are large and irregular they show no invasive tendency and do not invade the surrounding structure. In figure 4 showing a section of kidney in a case of lymphatic leukemia, there can be seen marked lymphocytic infiltrations which push aside and separate the tubules and glomeruli but do not invade and destroy them as occurs in malignant tumor. These infiltrations are accumulations of cells which resemble the picture produced by infection rather than the one produced by an invading tumor. It seems quite plausible that the various forms of leukemia and the diseases listed under blood dyscrasies have their origin in an infectious process. It is further conceivable that some body constituent or modification of the normal defense reaction in the body interferes with the normal activation of the manufacture and destruction of cells of the formative tissue.

The following diagram is presented to summarize these observations:



CONCLUSIONS

1. There is a constant physiological balance between the manufacture and destruction of blood cells; this process maintains an equilibrium which is compatible with health.

2. Introduction of a foreign substance, as for instance, infection, stimulates the formative tissue to greater production of blood cells; this may be called the normal producing capacity. Some abnormal body constituent may account for the lack of proper activation of these formative tissues, which results in some form of blood dyscrasia.

3. In the leukemias there is an hyperproducing capacity with over production of cells whereas in agranulocytosis there is an hypo-producing capacity with lack of cells. The histopathology of all leukemias indicates that there is no essential difference between the acute, chronic and aleukemic forms.

4. Many septic processes seem to produce the same clinical symptoms and histological changes that are seen in diseases of the hematopoetic system. Aplastic anemia, agranulocytosis and mononucleosis are usually associated with septic processes while Hodgkin's disease presents histological evidence of inflammation.

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GELATINOUS CARCINOMA OF THE BREAST

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Gelatinous carcinoma of the breast has recently been reviewed by Cheatle and Cutler,[2] who conclude that the origin of the gelatinous material is epithelial. That is to say, the gelatinous material is a product of degenerative changes within the tumor cells, or a product of their metabolism. The pathogenesis of this form of carcinoma of the breast has been the subject of some controversy. Cheatle and Cutler reviewed the literature, and their findings need not be repeated here. Suffice it to say that two opinions have been held. Some maintain that the gelatinous material is the result of stroma degeneration, and others maintain that the epithelial cells alone are responsible.

The opinion has been held, too, that these tumors are of a lower grade of malignancy than other carcinomas of the breast. Cheatle and Cutler maintain that they are as malignant as other carcinomas, and further point out that areas of gelatinous degeneration may be found in many forms of carcinoma of the breast. Recently, a case of gelatinous carcinoma of the breast was encountered in the laboratory, and because of its size and rather uniform histology, it was thought worthwhile to report it.

CASE REPORT

The tumor was obtained by operation from a woman thirty-nine years of age, who four years before had noticed a small nodule in the left breast. This nodule was quite hard, but not tender or painful. In the past year the tumor had grown very rapidly, so that at the time of operation the entire breast was involved in a hard, indurated tumor mass, the skin surface of which had ulcerated. Two months prior to the operation, a small nodule appeared in the right breast, and the left axilla was discovered to contain numerous hard glands. The tumor was now quite painful and obviously infected, at least superficially. In view of the nodule in the right breast and the axillary masses, radical operation was not performed, but only simple excision of the tumor in order to relieve the patient of pain and the complications of secondary infection.

The specimen (fig. 1) consisted of a breast occupied by a tumor measuring 10 by 8 by 6 cm. The skin was wrinkled and indurated, superficially ulcerated and slightly nodular. The cut surface revealed a diffuse growth of strikingly gelatinous appearance permeated by fine strans of stroma, giving an irregularly lobulated appearance. It was of pale gray color and resembled the cut surface of an indurated colloid goiter of considerable duration, except that it lacked the usual pink luster of a colloid goiter.

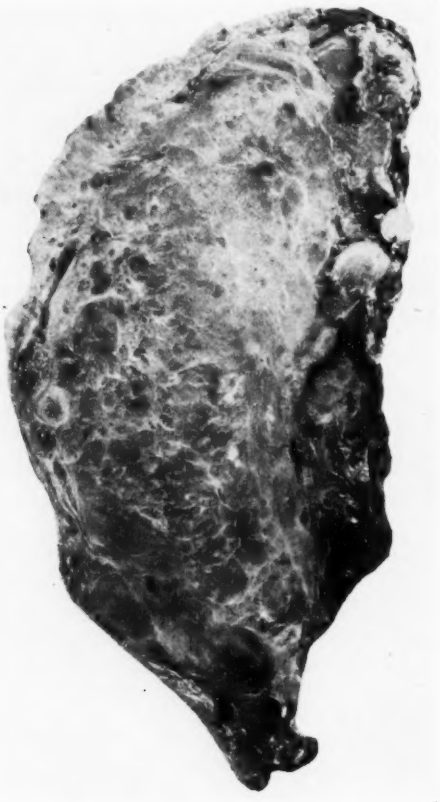

FIG. 1. GROSS SPECIMEN

Microscopic examination of a great many sections revealed a uniform histological picture. Coarse strands of acellular connective tissue made up the stroma. In places this stroma (fig. 2) was rather well preserved and took a deep eosin stain, whereas in other areas it stained poorly and seemed to have undergone degenerative change. The stroma was separated by masses of nonstaining material. At intervals, dilated ducts lined by flattened or slightly cuboidal and low columnar epithelium were present. Lying loosely in the gelatinous material

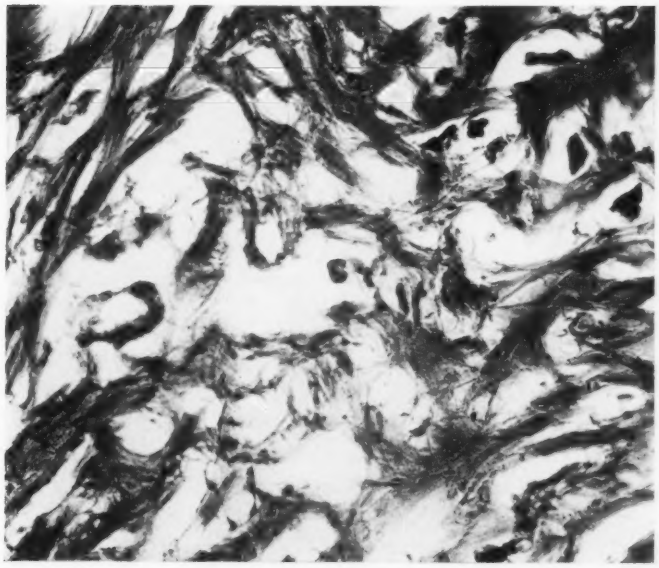

FIG. 2. PHOTOMICROGRAPH OF TUMOR
Note stroma

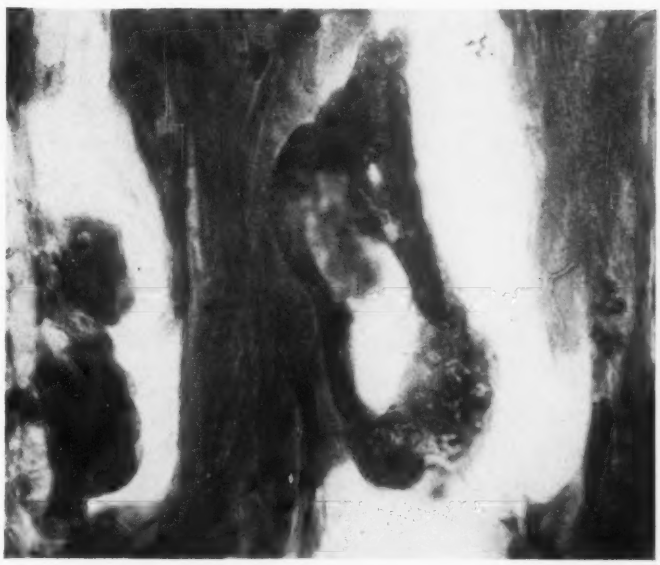

FIG. 3. PHOTOMICROGRAPH OF TUMOR
Note cytoplasmic vacuolization and nuclei crowded to one pole

were clusters of tumor cells. In the main, these cells maintained the appearance of small ducts or alveoli, but in places were arranged in solid nests. It was in these solid nests that the gelatinous material seemed to take origin. The cytoplasm of the cells was vacuolated. Frequently, the cells were massed together, forming a syncitial mass of tumor cells in which the cytoplasm had become vacuolated and the nuclei crowded together at one pole of the cell mass (fig. 3). The tumor cells were fairly uniform, with rather large oval and circular nuclei having a fine chromatin network and rather prominent nucleoli. Amitotic and mitotic division were occasionally noted. In some places the cells were so massed together as to have lost their outline, leaving only large numbers of nuclei imbedded in a mass of cytoplasm. It was frequently in these areas that the gelatinous formation seemed to be most active, although it occurred in even smaller groups of cells with only two and three nuclei. In some areas the cell cytoplasm had completely disappeared, leaving only shadowy remnants of the nuclei, which were in turn degenerating. Occasionally, extrusion of the vacuole seemed to be taking place, the vacuole bulging from the membrane of the cell. The sections from the deep portions of the tumor did not reveal any secondary inflammatory reaction, but the skin surface was the seat of a mild chronic inflammatory infiltration. In some of the ducts there was proliferation and piling up of the epithelium into the lumen. Here the cells were very large, the cytoplasm was vacuolated and closely resembled columnar cells of the colloid-secreting type. In some areas the syncitial masses of tumor cells were so filled with the gelatinous material that the cytoplasm was spread out in a thin shell, giving the appearance of minute cysts, with the remnants of the nuclei imbedded in the remnant of the cytoplasmic mass. Sometimes the nuclei were lying free in the vacuoles. In these areas there was lacking any evidence or else there was very scanty evidence of previous stroma. The stroma seemed to have been displaced and condensed to form denser bands, as described previously. A few tumor cells could be seen in what appeared to be lymphatic spaces, and occasional masses of atrophic tumor cells seemed to be imbedded in the denser stroma, giving an appearance suggestive of scirrhous cacinoma. Although the tumor was not richly cellular, the cells seemed to be vigorous and there was little evidence of spontaneous degeneration or necrosis.

Ewing mentioned that such tumors frequently metastasize as adenocarcinoma. Special staining of the tissue did not reveal any fat in the epithelium, and an extremely scanty amount in the stroma. The gelatinous areas stained with mucicarmen, and mucicarmen stained faintly, but not uniformly, some of the intracellular globules. It did not stain the intraductal secretion.

It would seem from this specimen that the conclusions of Cheatle and Cutler are supported. It is my opinion that the gelatinous material is a product of cell metabolism and not necessarily one of cell degeneration.

The patient survived for at least eight months after the operation. Since that time I have been unable to ascertain her condition. The history of the tumor is slightly less than five years. It seems reasonable to suppose that had the patient been operated upon when the tumor was first noted, cure might have been expected, since the slow development and the late appearance of metastases indicate a relatively benign form of carcinoma. From the sections it would seem that this tumor is of duct origin. The histology does not, in our opinion, necessarily indicate a low degree of malignancy, but since the tumor was four years old, it is quite possible that its primary cell form was different from that noted in the specimen.

Kaufman[4] was rather inclined to attribute the origin of the gelatinous substance to both degenerative changes in the stroma and secretory or degenerative changes in the epithelium. Ewing,[3] in four cases personally examined, was unable to attribute the origin of the gelatinous material to the epithelium. P. d'Allaines, Funck-Brentano and Pavie[1] reported an instance of colloid or gelatinous carcinoma of the breast occurring one year following the removal of a benign adenoma from the same breast. There was no evidence of any mucoid or gelatinous change in either the stroma or the epithelium of the benign adenoma. In the carcinoma, the gelatinous change was extensive, and there was evidence of the gelatinous substance in the lumen of the alveoli and in some of the terminal ducts. The authors were inclined to consider that the origin of the gelatinous material was in the stroma, but did not deny the possibility of primary epithelial change. Lazzarini[5] reported three cases of gelatinous carcinoma, and attributed the origin of the gelatinous material to the stroma. He was unable to find any evidence of primary change in the epithelium, and considered that the nutrition of the carcinoma cells was considerably interfered with by the degenerative changes in the stroma.

In the case reported here it seems important to note the goblet type of epithelium in some of the ducts, both in those which had undergone hyperplasia and in those which showed very little hyperplastic change.

Numerous sections taken from a considerable number of surgically removed breasts not involved by carcinoma and a

fewer number of samples of breast tissue removed at autopsy failed to reveal, either in the main ducts or in the deeper portions of the breasts, any goblet epithelium. The possibility that such epithelial cells may indicate an origin from sweat gland epithelium was considered, but we have been unable to detect any such epithelial changes in sections of sweat glands examined for a variety of lesions. It seems, therefore, that one is reduced to two possibilities to account for these goblet cells and the gelatinous secretion. One is degenerative change, for which there is considerable evidence, and the other is a functional and structural metaplasia. The former is the easier to accept, especially in view of the fact that areas of gelatinous change are frequently encountered in many types of carcinoma. However, the involvement of the entire tumor lends some support to the theory that this substance may not be the result of degenerative changes, but actually the result of a secretory function of the epithelium which has undergone metaplasia, or rather differentiation from the columnar epithelium to an actively secreting columnar epithelium. It is to be regretted that some of the metastases were not removed and examined, since it would be interesting to know if this tumor reproduced itself, and whether the metastases were of the adenocarcinoma type.

SUMMARY

An example of colloid or gelatinous carcinoma of the breast is described. The gelatinous material is interpreted as a product of the tumor cells.

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THE COLLOIDAL BENZOIN TEST OF CEREBROSPINAL FLUID

Its Clinical Value

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The colloidal benzoin test has been in use in the laboratory of the Los Angeles General Hospital for more than four years. This study is based on data collected from the first 2000 of these tests which were applied to routine spinal fluids from some 1800 patients. The study will be chiefly limited to the constancy and specificity of this reaction in syphilis of the central nervous system, poliomyelitis, tuberculous meningitis, epidemic encephalitis, and purulent meningitis. One may find complete references on the colloidal benzoin test in the excellent bibliography by Kermack and Voge.[8]

Keidel and Moore[7] in reporting the results from 311 cases considered the gum mastic test more delicate than the colloidal gold. Warnock[17] after studying eighty-seven cases was unfavorable toward benzoin and considered the gold test more reliable. Riddell and Stewart[15] from 100 tests were of the opinion that the benzoin was more delicate than the gold test. Wright and Kermack[19] found substantial agreement between the gold and benzoin tests and concluded that the latter was simpler and in many ways more satisfactory for clinical use. After studying the results from more than 1700 cases, Wassermann[18] considered the mastic equal to or more delicate than the gold test. From a series of 400 tests, Cockrill[1] was of the opinion that the mastic and benzoin tests were each of equal value to the gold test and that the benzoin test was simpler. Osborne,[10] after a comparative study of 1000 gold and benzoin tests, and of an additional 1000 benzoin tests alone, concluded that benzoin is superior to gold in ease of performance, and is more uniform, reliable, and

informative. Reyner[14] concluded that while the gold test is more informative than the mastic, although not more delicate, the benzoin test is the most delicate of the three, and more informative than the mastic test. Kermack and Voge[8] were still of the opinion that the benzoin test was easier to prepare, was simpler, and offered less likelihood of error and more likelihood of uniformity of results in routine laboratory work, than did the colloidal gold test.

The evidence therefore seems to favor the benzoin test as the simplest to prepare and read, as being as sensitive as and more informative than the mastic test and more sensitive than and as informative as the gold test.

TECHNIC

The original technic of Guillain and Léchelle[5] was used, as follows:

Glassware

Bottles, tubes (1 cm. by 6 cm.) 1 cc. and 10 cc. pipettes, all of nonsoluble glass. To be washed before using in 2 per cent aqueous hydrochloric acid and then twice in distilled water.

Solutions

Two solutions are needed: (1) a saline solution containing 0.1 gram of chemically pure sodium chlorid to 1000 cc. of twice distilled water; (2) a homogenous suspension of the resin of benzoin in distilled water, prepared as follows:

Dissolve 1 gram of the natural resin of benzoin (Sumatra) in 10 cc. absolute alcohol for forty-eight hours. The limpid liquid obtained is poured off and preserved as a stock solution. When a reaction is to be performed, 0.3 cc. of this alcoholic solution is added slowly to 20 cc. double distilled water heated to 35°C. in such a manner as to obtain a very homogeneous suspension. This must be freshly prepared and must not be kept over a couple of days. The water used must have been recently distilled.

Actual technic of reaction

Line up fifteen tubes with one extra for control.

To tube 1, add 0.25 cc. of the saline solution
To tube 2, add 0.50 cc. of the saline solution
To tube 3, add 1.50 cc. of the saline solution
To tubes 4 to 16 inclusive, 1 cc. of saline solution

Then pipet, shaking the mixture carefully after adding, the following amounts of spinal fluid:

To tube 1, 0.75 cc. spinal fluid
To tube 2, 0.50 cc. spinal fluid
To tube 3, 0.50 cc. spinal fluid

Then take 1 cc. of the contents of tube 3 and, having aspirated a few times to mix, transfer to tube 4. Mix the contents of tube 4 and transfer 1 cc. to tube 5. Continue this until tube 15 is reached, and reject 1 cc. from this tube, leaving tube 16 as a control.

Into each tube put 1 cc. of the benzoin suspension. The contents of all the tubes will be milky in appearance.

Place in ice-box and read in from six to twenty-four hours.

Interpretation

In the greater part of this series four numerals were used in recording the tests, namely, 0, 1, 2, and 3. A tube showing no precipitation was recorded as 0, a slight precipitation was called 1, almost complete precipitation but with cloudy supernatent fluid was designated by 2, while complete precipitation with clear supernatent fluid was read as 3. Three numerals only, 0, 1, and 2, were used in recording the earlier tests of the series; the 2 included both the later 2 and 3. In order to make the results uniform all 3's have been reduced to 2's throughout the series.

A normal reaction is usually indicated by a complete series of zeros; but as described by Guillain and Léchelle[5] there may normally be some precipitation in one or two tubes of numbers 5 to 8 inclusive. We have considered as normal precipitation less than grade 2 occurring in not more than three tubes in a zone.

Sources of error

Guillain et al.[3] repeated the colloidal benzoin test on cerebrospinal fluids kept at laboratory temperature for many days with no appreciable alteration in results. We have repeated the benzoin test on spinal fluids kept in the ice-box for one week with no appreciable difference in the curve providing the fluids remained sterile. It is probably best, however, to use spinal fluids not more than a few hours old to avoid the possibility of changes which may presumably alter the curve.

Schaffer[16] believed that precipitation of benzoin was due to variation in the hydrogen ion concentration of spinal fluid, or of the colloidal suspension. Papayanno[11] demonstrated the globulin factor in benzoin precipitation with spinal fluid.

Blood plasma in the spinal fluid will produce a curve in a normal fluid and alter a primary curve. The readings a, b, and c shown in table 1 were obtained by adding small amounts of the patient's blood plasma to the spinal fluid. The

plasma-free fluid used as a control gave a normal reaction with the benzoin. Bloody spinal taps probably constitute one of the greatest sources of error.

Because of its simplicity, technical errors are less likely to occur with the colloidal benzoin than with other similar "colloidal" tests and reading is dependent upon a precipitation rather than a change in color.

Zones

The fifteen tubes of the colloidal benzoin have been divided into two zones. The first zone comprises tubes 1 to 6, and the second zone includes tubes 7 to 15. The second zone is also called the tabetic and meningitic zone. The first zone has been called the paretic zone. Osborne found that a positive first zone reading in syphilis of the central nervous system usually meant general paresis, whereas in other forms of neurosyphilis the precipitation was confined to the second zone; also a positive first zone reading was considered to be an index of the degree of active involvement of the parenchyma of the brain, whereas purely vascular, meningeal, or spinal cord lesions produced precipitation in the second zone. Jaffrey[6] similarly concluded that there was this distinctive difference in zones in tabes and paresis, and that apparently the reaction was of value in finding where these two types of syphilis invade each other's territory.

A total of 2009 benzoin tests were run on fluids from 1800 cases, including 152 cases of syphilis of the central nervous system, 116 of tuberculous meningitis, 135 of poliomyelitis, nine of encephalitis, 316 of purulent meningitis, 616 miscellaneous diseases, and 456 cases in which the diagnosis was uncertain or complications were such as to make the case unsuitable for study.

SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

Of the 152 cases of syphilis of the central nervous system diagnosed clinically, all gave positive colloidal benzoin readings. The spinal fluid Wassermann was positive in 142 and negative in ten cases. The spinal fluid Wassermann had been positive in seven of the ten negative cases within the past three years and had become negative evidently as a result of treatment. The blood Wassermann was positive in the remaining three cases with negative spinal fluid Wassermann and no past records of spinal fluid examination were available.

In order to study the benzoin curves, all cases of this type of syphilis were divided into three groups according to the clinical diagnosis, namely general paresis, tabes dorsalis, and meningovascular syphilis.

General paresis. The colloidal benzoin test was applied to spinal fluids from seventy-six cases of paresis; thirty-eight cases were from our routine series and thirty-eight additional cases were obtained from the Norwalk State Hospital through the courtesy of Dr. Melvin J. Rowe of that staff. All cases of paresis were divided into two groups, those having received treatment for less than one year, and those having been treated for one year or more.

There were forty-seven cases in the first group and most of these were practically untreated. Forty-six or 97.9 per cent gave

TABLE 1

REPRESENTATIVE BENZOIN READINGS

Normal spinal fluid		000000000000000
Normal fluids—blood contamination	(a)	000001222222222
	(b)	222222000002222
	(c)	012222222222222
Average group curves		
General paresis (little or no treatment)		122222222221110
Tabes dorsalis		000001222221100
Meningo-vascular syphilis		000001222221000
General paresis (treated on average 4 years)		000001222110000
Tuberculous meningitis		000001222222100
Poliomyelitis		000000222110000
Epidemic encephalitis		000000000000000
Purulent meningitis		111111111211111
Three types of curve in purulent meningitis	(1)	000000222210000
	(2)	111222000222221
	(3)	111112222222222

a definite first zone reading, that is, precipitation occurred in three or more of the first six tubes, and in addition the curve extended to a varying degree into the second zone. All first zone readings were quite positive and for the most part the individual curve corresponded quite closely to the average curve for the group (table 1). Although all the readings extended into the second zone, the precipitation was as heavy in the paretic zone as in the succeeding zone and the curve was continuous without a drop in the middle. One case is of especial interest in that

the spinal fluid Wassermann had become negative after one year of treatment while the benzoin gave a typical paretic curve.

The twenty-nine cases comprising the second group of paretic patients had on an average received treatment over a period of four years. A positive second zone reading was obtained in each case but only six cases gave paretic zone readings. The average curve for the group is shown in table 1. The spinal fluid Wassermann was positive in all but five cases in which it had become negative during the period of treatment.

Tabes dorsalis. There were forty-three cases of tabes and each gave a positive second zone reaction in three or more tubes. Precipitation started in either tube 6 or 7 and the individual curve corresponded quite closely to the average for the group (table 1). In only two cases did the precipitation occur in three or more tubes in the paretic zone. Clinically, recent mental changes had been noted in each of these two patients. The spinal fluid Wassermann was positive in all but one case of the group, and in that one it had become negative during two years of treatment.

Meningovascular syphilis. This group included thirty-three cases each of which gave precipitation in three or more tubes in the second zone and corresponded in general to the readings in the tabetic group.

The paretic zone. In the entire series, including syphilis of the central nervous system there was but a single case, one of cerebellar tumor, in which precipitation was confined to the first zone. However, aside from these cases of syphilis and those of purulent meningitis, there were seventeen cases in the entire series having benzoin curves simulating curves in general paresis (table 2). These included one case each of cerebellar tumor, multiple sclerosis, tetanus, torula meningitis, four cases of coccidioides meningitis; and nine cases of tuberculous meningitis. Guillain et al.[4] in reporting the results of the colloidal benzoin on spinal fluids from twenty-eight patients with cerebral tumor, found no curves of the paretic type; the benzoin was negative in five cases, precipitation was present in the second zone only in seventeen cases, and was present in both the first and second zones with negative tubes between the zones in six cases. Neither is the paretic curve

the rule in tetanus since in our series there were twenty cases, the curve in only one of which somewhat simulated paresis. A case of multiple sclerosis was one out of three. The nine cases of tuberculous meningitis were from a group of 116 cases. The four cases of meningitis due to coccidioides and the one case of torula meningitis represented all of the cases of these diseases.

TABLE 2

CASES, NOT SYPHILIS, WITH PARETIC TYPE CURVE

NUMBER	CURVE	DIAGNOSIS
1	210000000000000	Cerebellar tumor
2	002002222200000	Multiple sclerosis
3	111211222211111	Tetanus
4	002222112222210	Tuberculous meningitis
5	112212222222222	Tuberculous meningitis
6	222202222222222	Tuberculous meningitis
7	122222222222222	Tuberculous meningitis
8	022222222222000	Tuberculous meningitis
9	222222202222222	Tuberculous meningitis
10	000122222222100	Tuberculous meningitis
11	012222222222222	Tuberculous meningitis
12	222202222222222	Tuberculous meningitis
13	122222222222210	Coccidioides meningitis
14	000222212222222	Coccidioides meningitis
15	222202222211000	Coccidioides meningitis
16	002222222222222	Coccidioides meningitis
17	000002222222221 012222222222222	Torula meningitis

TUBERCULOUS MENINGITIS, POLIOMYELITIS, AND EPIDEMIC ENCEPHALITIS

These diseases will be considered in one group because of their clinical relationship, and because in certain instances there is difficulty in the differential diagnosis.

Tuberculous meningitis. Benzoin tests were run on specimens of spinal fluid from 116 patients. A positive reading was obtained in 97.4 per cent. Three cases in which the spinal fluid findings were otherwise consistent with the clinical diagnosis gave a normal benzoin reaction, and although the tubercle bacillus was not found in the spinal fluid, these cases were clinically

diagnosed as tuberculous meningitis and went on to a fatal termination. The average curve for the series is shown in table 1 and occupies the second zone. Readings in eleven cases gave curves simulating that found in cases of general paresis in that heavy precipitation started in the first zone, involved three or more tubes, and continued uninterrupted into the second zone. Two of these cases had a positive spinal fluid Wassermann. The remaining nine are charted in table 2. They were all terminal cases at the time the spinal fluid was taken.

Poliomyelitis. This group included 135 cases of poliomyelitis in which the colloidal benzoin test was applied to the spinal fluid within the first two weeks of the disease. Ninety-four and seven-tenths per cent gave a positive reaction in the second zone. There were no first zone reactions. Occasionally a negative reading was obtained on the first or second day of the disease, but when the test was repeated the following day the result was a typical curve. The individual curves were quite uniform, and in the main corresponded very well to the average curve for the group (table 1).

In seven cases the benzoin test was normal; the spinal fluid was taken sometime between the first and seventh day of the disease, and examination of the spinal fluid was otherwise consistent with the clinical diagnosis. Paralysis was present in five of the seven cases.

These results are quite similar to those of Regan[13] who obtained a positive reaction in twenty-two of twenty-three cases of poliomyelitis and the zone of reaction was in tubes 6 to 9, often extended to 10, and occasionally to 11 and 12. He found negative tests were of increasing frequency after the fourth week of the disease, and spinal fluids in the sixth week were normal.

Encephalitis. There were nine cases, four of epidemic encephalitis, two of encephalitis following influenza, two of encephalitis complicating measles, and one case of the Marie Strümpell type. The spinal fluid from each of these patients gave a normal benzoin reaction except that from a patient having complicated measles by encephalitis, in which a positive second zone reaction was obtained.

Guillian and Léchelle reported three cases of epidemic encephalitis and a fourth post-encephalitic case in each of which the colloidal benzoin reaction was normal. Six cases of epidemic encephalitis were reported by Duhot and Crampton,[2] and ten cases by Rabeau,[12] in all of which the colloidal benzoin was negative. Ménard[9] found the benzoin constantly negative in epidemic encephalitis and positive in tuberculous meningitis.

Whereas the average curve of the group in poliomyelitis differs somewhat from the average curve of the group in tuberculous meningitis, the individual curve in the one condition may simulate the average curve of the other in many instances and therefore it is questionable if the benzoin test is of value in differentiating between these two conditions. However, there is evidence that the colloidal benzoin test may be of considerable value in the differentiation of epidemic encephalitis from poliomyelitis and tuberculous meningitis.

PURULENT MENINGITIS

The colloidal benzoin test was applied to the specimens of spinal fluid from 316 patients having purulent meningitis. There were 221 cases of the epidemic type and ninety-five miscellaneous cases of which the pneumococcus was the most frequent cause. The test was positive in 99 per cent of cases. The precipitation was not confined to one zone although heavier in the second zone. The individual curve did not correspond very closely to the average curve for the group which involved all fifteen tubes (table 1). Early in the disease however, the reading was usually confined to the second zone. Later precipitation extended to the end of the second zone, and forward into the first zone. Not infrequently a double curve resulted with precipitation in practically all tubes except three or four in the middle. The various types of purulent meningitis were indistinguishable as far as the colloidal benzoin reaction was concerned.

MISCELLANEOUS GROUP

This group comprised 616 cases, 150 of which were diseases of the central nervous system. In general, fluid from patients with

organic disease of the central nervous system gave positive readings confined to the second zone with the exception of those cases discussed in relation with the paretic zone. The group included cases of multiple sclerosis, subacute combined sclerosis, bulbar palsy, cerebral hemorrhage and thrombosis, epilepsy, rabies, botulism, tetanus, psychoses, et cetera.

In the great majority of miscellaneous diseases other than those of the central nervous system there was a normal benzoin reaction, as might be expected. However, it was found that certain types of disease were apt to have a second zone curve, namely: infectious diseases such as lobar pneumonia accompanied by meningismus, diseases such as diabetes mellitus and nephritis, accompanied by acidosis or nitrogen retention, and acute inflammatory processes adjacent to the dura mater such as sphenoid sinusitis, and mastoiditis in which the spinal fluid shows no bacteria, although there may be a slight increase in lymphocytes and globulin.

SUMMARY

(1) The evidence in the literature indicates that of the colloidal tests used in examination of the spinal fluid the benzoin test is the simplest to prepare and read, is as sensitive as and more informative than the gum mastic test, and is more sensitive than and as informative as the colloidal gold test.

(2) A study of the data obtained from some 2000 colloidal benzoin tests on cerebrospinal fluids from 1800 patients signifies that: (a) the benzoin test is not a specific test in the same sense as the Wassermann reaction, (b) it is of value in differentiating active general paresis from other forms of neurosyphilis, (c) it is probably of value in differentiating epidemic encephalitis from poliomyelitis and tuberculous meningitis, (d) a high percentage of positive readings is obtained in disease of the central nervous system, but a positive second zone reading is occasionally obtained in certain conditions not associated with organic disease of the central nervous system.

We are indebted to Dr. Charles Dale for his help in the accumulation of these data, and to Miss Bertha Gannon for the technical work.

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EDITORIAL

Death Certificate Diagnoses

The importance of accurate mortality statistics to the health of a nation is fully appreciated by the educated layman as well as the physician. That the progress of medicine is dependent in a not insignificant measure on the accuracy of such statistics is a fact not so fully appreciated even by physicians, upon whom the responsibility for the accuracy of the figures depends.

Clinical diagnoses are seldom correct in their entirety even when made by physicians whose ability is unquestioned. When there is added to the excusable error of judgment a certain amount of carelessness and lack of knowledge of the principles of classification for mortality statistics the error becomes enormous. The result is that instead of mortality tables being helpful they may do harm because they are misleading. So great a factor in the public welfare and the progress of medicine deserves more than ordinary effort toward accuracy on the part of those who are responsible for the diagnoses written on the death certificates since these are the source from which mortality tables are derived.

Every pathologist of experience has witnessed the uncertainty, even confusion of the clinician in deciding upon the cause of death to be written on the death certificate. Too often the pathologist is content to write his anatomical diagnosis and watch in silence the efforts of his colleague to translate or qualify this anatomical diagnosis so that it will be acceptable to the registrar of his state.

For evidence that the difficulties of the physician are real the figures of Wood may be cited. He states that in Pennsylvania one in every forty death certificates is so incomplete as to require correspondence with the physician and that every year 4500 physicians are asked to correct their diagnoses or supply deficiencies. The chief reason for these difficulties of the physician

lies in the difference of his point of view from that of the public health official.

The physician is trained to think of disease as abnormality of function and structure and makes his diagnosis on this basis whereas a satisfactory diagnosis from point of view of the public health official must take into consideration the factors responsible for bringing about these abnormalities of function and structure.

It is for this reason that the diagnosis of bronchopneumonia not qualified by the adjective "primary" is so often returned for additional information. The registrar knows that bronchopneumonia is more often a terminal complication of some other condition and it is the primary disease that he wants. If bronchopneumonia follows an acute infectious disease such as measles or whooping cough he is more interested in the possibility of forestalling an epidemic than in the anatomical and functional cause of death—consolidation of the lungs by an exudate caused by a toxin producing streptococcus. Similarly a diagnosis of fracture of the skull with laceration of the brain will satisfy the pathologist and keeper of hospital records but the keeper of mortality records for the state must know the cause of the fracture, not only the manner in which it was brought about but whether it was accident, suicide, or homicide. Again the idea of prevention is uppermost in his mind.

Pathologists can perform a real service if they will study the principles underlying the classification for general mortality statistics and familiarize themselves with the International List of the Causes of Death as adopted by the greater part of the civilized world including the Bureaus of Vital Statistics in the Registration Area of the United States. By doing this they will be in a position to assist the physician in what is at times a difficult task, increase the accuracy of important figures, and incidentally make additional friends.

W. S. Thomas.

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The following will act as a program committee for the Twelfth Annual Convention of the A. S. C. P.: A. S. Giordano, Chairman, A. H. Sanford and F. W. Hartman. The local committee at Milwaukee will consist of Norbert Enzer, Chairman, E. L. Tharinger, E. F. Barta and Marcos Fernan-Nunez.

During the absence of Dr. H. C. Schmeisser, Dr. M. Pinson Neal, professor of Pathology at the University of Missouri School of Medicine, has served as visiting professor in the Department of Pathology at the University of Tennessee College of Medicine and as acting director of the laboratories of the Memphis General Hospital.

Dr. John A. Kolmer has been appointed professor of Medicine at Temple University.

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BOOK REVIEWS

The Chemistry of Tuberculosis. 2nd Edition. By H. Gideon Wells and Esmond R. Long. Pp. xiv + 481, 1932, Baltimore, The Williams & Wilkins Company, $7.00.

This well known work which is essentially a review of literature, has been thoroughly rewritten to include the newer views concerning the chemistry of tuberculosis. The book has been enlarged by one new chapter and thirty-two pages of text. But this does not indicate the extent of the changes, for one notes that whereas the original text contained about 1100 references to literature, the present edition contains nearly 1700 which indicates that about seventy-five contributions have appeared every year since the first edition. In the chapter on the growth and metabolism of *M. tuberculosis*, more than 100 new references appear. The book represents an excellent summary of all that is known of the chemistry of this disease and its causative organism. The chapters are not only summarized, but the contents of each are critically reviewed in a few brief paragraphs.

Human Cancer. By Arthur P. Stout. Pp. viii + 1007, 1932, Philadelphia, Lea & Febiger, $10.00.

This monograph approaches the subject of cancer from a different aspect than most texts on the subject. The author has presented his material by regions of the body, discussing the malignant growths of each region in relation to etiological factors, precancerous lesions, growth, spread, symptoms, diagnosis, prognosis and principles of treatment. He has accepted the factor of chronic irritation as a working hypothesis for the cause of cancer which he discusses at length. Most of the highly speculative phases of the subject are omitted as well as academic experimental cancer research. A well chosen bibliography at the end of each chapter, records the literature by years during the past quarter century or more, the author, with rare honesty admitting that

it is not complete and that he has not read it all. The illustrations, numbering more than 300, are original and excellent. An appendix by G. F. Laidlaw gives the technic for silver staining.

The book exhibits a great amount of labor and intelligence on the part of the author and makes a practical, yet extensive, modern treatise on cancer, useful not only to the surgeon and pathologist, but to the internist and radiologist.

Quantitative Clinical Chemistry. Volume II, Methods. By John P. Peters and Donald D. Van Slyke. Pp. xx + 957, 1932, Baltimore, The Williams & Wilkins Company, $10.00.

This is a fitting companion to the previously published volume on "Interpretations." In it one finds the exact steps, given in detail, for carrying out the tests, the results of which were discussed in the first volume of this monumental work.

The book contains a clear exposition of the use of the equipment needed to perform quantitative clinical chemical tests and includes the answers to hundreds of technical questions that arise in the minds of those performing biochemical procedures. As would be expected the authors devote a generous share of the work to gasometric methods on which they are leading authorities. But for the most part at least one gravimetric, titrimetric and colorimetric method is given for the determination of each substance. The style of presentation is excellent, direct and clear; each test is described by giving the apparatus required, the reagents, the procedures and the exact method of calculation to obtain the result. Illustrations, tables, nomograms and references are plentiful.

It is unfortunate that so excellent a text should have so poor an index, for many items would never be suspected of being present were one to consult the index alone. The same condition also prevails with reference to the author index. There are some omissions in the text also as for example tests for enzymes, carotin, certain heavy metals such as lead and arsenic, nitrates and nitrites, bile salts and acids, and there is no discussion of sugar tolerance tests and the Congo red method for blood volume determinations. There is an evident error in the description of the diazo reaction where the color is described as being blue. But these omissions seem trivial when the book is considered as a whole.

American Journal of Clinical Pathology

Manuscripts and books for review should be sent to Dr. Thomas B. Magath, Mayo Clinic, Rochester, Minnesota. Manuscripts must be typewritten and all figures and tables should be in such form as to be ready for the printer. The expense for a limited number of cuts can be borne by the Society; expense for cuts in excess of this number will have to be defrayed by the author. The nomenclature for species of bacteria will be that given in Bergey's "Manual of Determinative Bacteriology." Bibliographic references will be limited to the papers actually referred to in the text. Such citations must be arranged in alphabetic sequence and made in the following form: author's name followed by initials, title, journal, volume, inclusive pages, date.

(Examples) Kolmer, J. A.: Toxin production by *Spirochaeta pallida*. Arch. Derm. and Syph., 20: 189-196. 1929.

McFarland, Joseph: A text-book upon the pathogenic bacteria and protozoa for students of medicine and physicians. Philadelphia and London: W. B. Saunders Company, 1919, pp. 853.

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